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Aggregational Capacity Of Platelets In Patients With Dyslipidemia.

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ABSTRACT

Previous studies have shown that among the population of industrially developed countries there is a high prevalence of dyslipidemia. It is very dangerous for the development of thrombosis of blood vessels in this category of patients. They are based on the hyperaggregation of blood cells, the severity of which has recently been gradually calculated. The goal is to evaluate the aggregation capacity of platelets in patients with dyslipidemia. We examined 380 patients of the second adult age (mean age 53.8 ± 1.6 years) with dyslipidemia type IIb. The control group consisted of 26 clinically healthy people of the same age. All the examined persons gave written informed consent to participate in the study. Biochemical, hematological and statistical methods of investigation were used in the work. As a result of the study, it became clear that a high incidence of thrombosis of various localizations with dyslipidemia is closely related to the development of platelet hyperaggregation. This phenomenon is based on the weakening of the antioxidant protection of the plasma with the activation of the processes of lipid peroxidation in it. The study also found that people with dyslipidemia have a weakened ability of platelets to disaggregate. As a result, patients have a sharply increased risk of thrombosis of any location, which can lead to disability and death.

Keywords: platelets, dislipidemia, vascular wall, antiaggregation.

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INTRODUCTION

A steady improvement in the quality of life of the population in many countries of the world leads to a widespread prevalence of excessive diet in them, leading to dyslipidemia [1,2]. This leads to an increase in the incidence of vascular, often lethal thromboses among persons of working age [3]. At the heart of this in this contingent of patients often lies the hyperaggregation of blood cells [4]. Its important manifestation is the stimulation of hemostasis processes with the development of thrombophilia [5,6,7]. This is due to the weakening of the sensitivity of blood cells to disaggregants [8,9]. Given the wide prevalence of dyslipidemia, it is of great interest to study the level of platelet aggregation in this contingent of patients [10].

The aim of the study is to evaluate the aggregation capacity of thrombocytes in patients with dyslipidemia.

MATERIAL AND METHODS

The research was approved by the Ethics Committee of Kursk Institute of Social Education (branch of Russian State Social University) (record №5 from 12.05.2014).

We examined 41 patients of the second mature age (mean age 53.8±1.6 years) with dislipidemia of IIb type [11]. The control group was composed of 26 clinically healthy people of the same age. All the examined persons gave written informed consent on participation in the research.

We determined the content of common cholesterol (CS) and triglycerides (TG) in blood of all the observed persons by enzymatic colorimetric method with the help of a kit “Vital Diagnostikum” (Russia). CS level of high-density lipoproteins (HDLP) was determined with the help of a kit “Olveks Diagnostikum (Russia) by enzymatic colorimetric method. Common lipids (CL) were estimated with the help of a kit “Erba Russ” (Russia). The quantity of common phospholipids (CPL) in blood plasma was registered according to the content of phosphorus in them. CS levels of low-density lipoproteins (LDLP) were established by calculation according to Freedwald V. CS concentrations of very low-density lipoproteins (VLDLP) was determined according to the formula: TG content/2.2. Received indices of common CS and CS of LDLP were considered as normal, border-line or high in accordance with Russian recommendations (2012) [12].

Intensity of lipids’ peroxidation (LPO) processes in plasma was estimated according to the content of thiobarbituric acid (TBA)-active products by a kit “Agat-Med” and acylhydroperoxides (AHP) [13]. Antioxidant abilities of liquid part of blood were determined according to the level of its antioxidant activity (AOA) [14].

LPO activity in studied regular blood elements was determined according to the quantity of malon dialdehyde (MDA) in reduction reaction of thiobarbituric acid in washed and resuspended cells and the content of AHP in them [13]. In studied washed and resuspended regular blood elements we estimated the levels of cholesterol by enzymatic colorimetric method with the help of a kit “Vital Diagnostikum” and CPL according to the content of phosphorus in them.

Aggregation of platelets (AP) was assessed by visual micromethod [15] using ADP ($0.5x10^{-4}$ M), collagen (1:2 dilution of the base suspension), thrombin (0.125 ed/ml), ristomycin (0.8 mg/ml), epinephrine ($5.0×10^{-6}$ M) and with combinations of ADP and epinephrine; ADP and collagen; epinephrine and collagen at the same concentrations in a platelet-rich plasma with a standardized platelet count of $200×10^9$ platelets. Aggregational capacity of platelets inside the vessels was determined using a phase contrast microscope [16]. Considered the number of small, medium and large aggregates and the involvement of platelets in them [17,18].

The results were processed by Student’s criterion (t). Statistical processing of received information was made with the help of a program package “Statistics for Windows v. 6.0”, “Microsoft Excel”. Differences in data were considered reliable in case of $p<0.05$.

RESEARCH RESULTS AND DISCUSSION

The blood of patients was noted to have levels’ increase of CL and common CS which surpassed the control values in 1.6 and 1.3 times, respectively, at simultaneous lowering of plasma CPL in 2.1 times (Table).

The blood of persons with dislipidemia was found to have the increase of CS LDLP, CS VLDLP and TG in 1.64, 1.61 and 1.60 times, respectively. It is combined with the lowering of CS HDLP in 1.55 times. The patients were noted to have evident plasma LPO activation – the content of AHP in it surpassed the control value in 2.1 times, TBA-active products – in 1.4 times, being accompanied by suppression of antioxidant plasma activity in 1.3 times (Table).

The observed patients were noted to have increased CS content in erythrocytes' membranes which was accompanied by the decrease of CPL in them and LPO activation on behalf of weakening of their antioxidant protection (Table).

Table. Registered indicators in the surveyed

Registrated parameters	**Patients with arterial hypertension and dyslipidemia, n=41, M±m**	**Control, n=26, M±m**
common cholesterol, mmol/l	6.3±0.05	4.8±0.05 p<0.01
CS level of high-density lipoproteins, mmol/l	1.07±0.06	1.60±0.06 p<0.01
CS levels of low-density lipoproteins, mmol/l	3.99±0.07	2.43±0.04 p<0.01
CS concentrations of very low-density lipoproteins, mmol/l	1.24±0.04	0.77±0.05 p<0.01
triglycerides, mmol/l	2.72±0.06	1.70±0.02 p<0.01
common lipids, g/l	9.0±0.10	5.6±0.03 p<0.01
common phospholipids, mmol/l	1.70±0.06	3.54±0.09 p<0.01
acylhydroperoxides plasma, D_{233} /1ml	3.01±0.07	1.42±0.09 p<0.01
TBA-compounds, µmol/l	4.92±0.08	3.56±0.07 p<0,01
antioxidant activity plasma, %	24.2±0.13	32.9±0.12 p<0.01
biochemical parameters of platelets		
cholesterol of platelets, µmol/10^9 platelets	1.02±0.006	0.67±0.005 p<0.01
common phospholipids of platelets, µmol/10^9 platelets	0.34±0.003	0.49±0.004 p<0.01
acylhydroperoxides of platelets, D_{233}/10^9 platelets	3.06±0.04	2.20±0.04 p<0.01
malonic dialdehyde of platelets, nmol/10^9 platelets	1.28±0.07	0.68±0.02 p<0.01
catalase of platelets, ME/10^9 platelets	5350.0±17.05	9790.0±20.10 p<0.01
superoxidismutase of platelets, ME/10^9 platelets	1200.0±6.30	1650.0±3.00 p<0.01
aggregation of platelets		
aggregation with ADP, s	25.3±0.08	41.0±0.12

		p<0.01
aggregation with collagen, s	23.4±0.10	33.2±0.10 p<0.01
aggregation with thrombin, s	36.7±0.13	55.3±0.05 p<0.01
aggregation with ristomycin, s	28.7±0.10	45.2±0.06 p<0.01
aggregation with epinephrine, s	72.4±0.14	93.0±0.07 p<0.01
aggregation with ADP and epinephrine, s	21.3±0.16	34.5±0.04 p<0.01
aggregation with ADP and collagen, s	18.5±0.12	26.6±0.05 p<0.01
aggregation with epinephrine and collagen, s	16.2±0.11	29.2±0.12 p<0.01
The number of platelets in the aggregates, %	10.0±0.14	6.5±0.07 p<0.01
Number of little aggregates (in 100 free thrombocytes)	11.2±0.10	3.1±0.03 p<0.01
Number of medium and large aggregates (in 100 free thrombocytes)	1.28±0.09	0.14±0.03 p<0.01

Note: p - reliability of differences in the indices of a group of patients and a control group.

In patients with dyslipidemia, acceleration of development of AP with individual inducers and their combinations was found (Table). The earliest time the AP came under the action of collagen, a little later with ADP, even later with ristomycin, thrombin and adrenaline. The AP in response to a combination of inductors was also accelerated. The number of free-circulating patients with platelet aggregates of different sizes and the degree of platelet involvement in them in persons with dyslipidemia significantly exceeded the control values.

Important significance in the development of rheological disturbances and thrombophilia in persons with dislipidemia belongs to aggregation increase of regular blood elements and especially – platelets [19,20]. At dislipidemia the depression of plasma antioxidant activity is formed which provides the increase of LPO activity in it [21,22]. The increase of freely radical processes in liquid part of blood inevitably promotes the damage of platelets' membranes. The development of these manifestations in combination with found in these patients' platelets lipid imbalance leads to their hyperaggregability. At the same time, platelet levels lower the level of disaggregation [23,24].

Amplification of AP with individual inducers and their combinations is based on simultaneous enhancement of aggregation mechanisms and attenuation of disaggregation properties [25,26]. Apparently, this is caused by dyslipidemia and activation of LPO in plasma [27,28]. Acceleration of AP in response to ristomycin in patients is associated with an increase in the number of receptors for von Willebrand factor on platelets [29,30]. Acceleration of AP in response to combinations of inducers and an excessive number of aggregates of platelets in the blood of patients indicated a marked increase in platelet aggregation mechanisms [31, 32].

CONCLUSION

The high frequency of thrombosis of various localizations with dyslipidemia is largely due to the formation of platelet hyperaggregation. In conditions of dyslipidemia, weakening of the antioxidant protection of the plasma with activation of the LPO processes in it, leading to changes in platelet activity, is noted. It was found that persons with dyslipidemia have an obvious weakening of the ability to disaggregate in platelets. As

a result, patients receive a sharply increased risk of thrombosis of any location, which can lead to disability and death [33,34,35].

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